

# WEST Search History

DATE: Tuesday, July 16, 2002

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
	<i>DB=USPT; PLUR=YES; OP=AND</i>		
L37	l33 and l36	0	L37
L36	g adj protein adj fusion adj protein	5	L36
L35	l33 and l34	95	L35
L34	fusion adj protein	12987	L34
L33	calcium and glutamate and gaba	480	L33
L32	l30 and l31	0	L32
L31	(g adj protein adj fusion)	10	L31
L30	l24 or l25 or l29	4	L30
L29	l19 and l23	3	L29
L28	l15 and l23	0	L28
L27	l12 and l23	0	L27
L26	l8 and l23	0	L26
L25	l5 and l23	4	L25
L24	l2 and l23	3	L24
L23	l20 and l22	363	L23
L22	fusion or chimera	67154	L22
L21	l2 and l20	3	L21
L20	gaba	2167	L20
L19	l17 and l18	3	L19
L18	rachel.in.	359	L18
L17	simin.in.	13	L17
L16	simin.in.l15	0	L16
L15	l13 and l14	78	L15
L14	james.in.	119253	L14
L13	garrett.in.	1100	L13
L12	james.in. and l11	33	L12
L11	busby.in.	129	L11
L10	l4 and l9	4	L10
L9	lance.in.	1354	L9
L8	l6 and l7	0	L8
L7	laura.in.	1353	L7

L6	storjohann.in.	4	L6
L5	lance and L4	4	L5
L4	hammerland.in.	4	L4
L3	hammeland.in.	0	L3
L2	thomas.in. and 11	5	L2
L1	stormann.in.	7	L1

END OF SEARCH HISTORY

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 17:10:26 ON 16 JUL 2002

=> file biosis caplus medline

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'BIOSIS' ENTERED AT 17:10:35 ON 16 JUL 2002

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FILE 'CAPLUS' ENTERED AT 17:10:35 ON 16 JUL 2002

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FILE 'MEDLINE' ENTERED AT 17:10:35 ON 16 JUL 2002

=> stormann?/au

L1 138 STORMANN?/AU

=> thomas?/au

L2 132992 THOMAS?/AU

=> l1 and l2

L3 0 L1 AND L2

=> hammerland?/au

L4 56 HAMMERLAND?/AU

=> lance?/au

L5 4934 LANCE?/AU

=> l4 and l5

L6 0 L4 AND L5

=> storjohann?/au

L7 38 STORJOHANN?/AU

=> laura?/au

L8 1424 LAURA?/AU

=> l7 and l8

L9 0 L7 AND L8

=> busby?/au

L10 2147 BUSBY?/AU

=> james?/au  
L11 47000 JAMES?/AU  
=> l10 and l11  
L12 4 L10 AND L11  
=> garrett?/au  
L13 11267 GARRETT?/AU  
=> l11 and l13  
L14 27 L11 AND L13  
=> simin?/au  
L15 2057 SIMIN?/AU  
=> rachel?/au  
L16 751 RACHEL?/AU  
=> l15 and l16  
L17 0 L15 AND L16  
=> gaba  
L18 97789 GABA  
=> fusion or chimera?  
L19 455775 FUSION OR CHIMER?  
=> l12 and l19  
L20 0 L12 AND L19  
=> l14 and l19  
L21 0 L14 AND L19  
=> gaba or glutamate or calcium  
L22 1485083 GABA OR GLUTAMATE OR CALCIUM  
=> l19 and l22  
L23 15763 L19 AND L22  
=> g protein(s)fusion  
L24 1769 G PROTEIN(S) FUSION  
=> g protein(s)chimera?  
L25 1327 G PROTEIN(S) CHIMER?

=> 124 or 125

L26 2886 L24 OR L25

=> 122 and 126

L27 428 L22 AND L26

=> 127 and 1970-2000/py

L28 311 L27 AND 1970-2000/PY

=> g protein fusion protein

L29 33 G PROTEIN FUSION PROTEIN

=> g protein chimera?

L30 30 G PROTEIN CHIMER?

=> 129 or 130

L31 61 L29 OR L30

=> 122 and 131

L32 9 L22 AND L31

=> 132 and 1970-2000/py

L33 4 L32 AND 1970-2000/PY

=> dup rem 133

PROCESSING COMPLETED FOR L33

L34 4 DUP REM L33 (0 DUPLICATES REMOVED)

=> d ti abs so 134 1-4

L34 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2002 ACS

TI Gonadotropin-releasing hormone receptor initiates multiple signaling pathways by exclusively coupling to Gq/11 proteins

AB The agonist-bound gonadotropin-releasing hormone (GnRH) receptor engages several distinct signaling cascades, and it has recently been proposed that coupling of a single type of receptor to multiple G proteins (Gq,

Gs, and Gi) is responsible for this behavior. GnRH-dependent signaling was studied in gonadotropic .alpha.T3-1 cells endogenously expressing the murine receptor and in CHO-K1 (CHO#3) and COS-7 cells transfected with the

human GnRH receptor cDNA. In all cell systems studied, GnRH-induced phospholipase C activation and Ca<sup>2+</sup> mobilization was pertussis toxin-insensitive, as was GnRH-mediated extracellular signal-regulated kinase activation. Whereas the Gi-coupled m2 muscarinic receptor interacted with a chimeric Gs protein (Gsi5) contg. the C-terminal five amino acids of G.alpha.i2, the human GnRH receptor was unable to activate the **G protein chimera**. GnRH challenge of .alpha.T3-1, CHO#3 and of GnRH receptor-expressing COS-7 cells did not result in agonist-dependent cAMP formation. GnRH challenge of CHO#3

cells

expressing a cAMP-responsive element-driven firefly luciferase did not result in increased reporter gene expression. However, coexpression of the human GnRH receptor and adenylyl cyclase I in COS-7 cells led to clearly discernible GnRH-dependent cAMP formation subsequent to GnRH-elicited rises in  $[Ca^{2+}]_i$ . In  $\alpha$ .T3-1 and CHO#3 cell membranes, addn. of  $[\alpha\text{-}^{32}P]\text{GTP}$  azidoanilide resulted in GnRH

receptor-dependent

labeling of G $\alpha$ .q/11 but not of G $\alpha$ .i, G $\alpha$ .s or G $\alpha$ .12/13 proteins. Thus, the murine and human GnRH receptors exclusively couple

to

G proteins of the Gq/11 family. Multiple GnRH-dependent signaling pathways are therefore initiated downstream of the receptor/G protein interface and are not indicative of a multiple G protein coupling potential of the GnRH receptor.

SO Journal of Biological Chemistry (2000), 275(13), 9193-9200  
CODEN: JBCHA3; ISSN: 0021-9258

L34 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI Regulation of neuronal signaling pathways by chemokines and gpl20.

AB We have recently demonstrated that diverse chemokine receptors including CXCR4 and CCR5 are expressed by many kinds of neurons, including dorsal root ganglion(DRG) and hippocampal neurons. We further studied the consequences of chemokine receptor activation in neurons and HEK 293 cells. Chemokine receptors were expressed using PEI-mediated transfection together with CD8 or GFP as markers. MDC, RANTES, SDF-1 $\alpha$  and fractalkine inhibited I $Ca$  in a voltage dependent and NEM/pertussis toxin sensitive manner in rat(r) CCR4, rCCR5, rCXCR4 and rCX3CR1 expressing HEK 293 cells stably expressing N-type **calcium** channels. SDF-1 $\alpha$  and fractalkine mobilized  $(Ca^{2+})_i$  in HEK293 cells when their receptors were expressed together with Galphai5, a **G-protein chimera** which switches the Gi coupled receptors to the Gq mediated pathway. gpl20IIIB, which uses CXCR4 as a coreceptor, did not inhibit I $Ca$  or cause **calcium** mobilization in rCXCR4 expressing cells unless the human(h) CD4 molecules was coexpressed. gpl20IIIB also produced much greater internalization of GFP-tagged rCXCR4 expressed in HEK293 cells if hCD4 was coexpressed. GFP-tagged rCXCR4 expressed in cultured rat hippocampal pyramidal neurons were generally internalized suggesting that they were mostly already down-regulated possibly by SDF-1 $\alpha$  released from the glial feeder layers. These results show that gpl20IIIB can utilize rCXCR4 as coreceptors and can activate these receptors if hCD4 is also expressed. The ability of gpl20IIIB to produce effects in rat neurons

suggest that they express CXCR4 and another coreceptor that can take the place of hCD4.

SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-606.5. print.  
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience  
. ISSN: 0190-5295.

L34 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI Expression of functional **GABA-B** receptors in CHO cells containing chimeric and promiscuous G proteins.

SO Society for Neuroscience Abstracts., (1999) Vol. 25, No. 1-2, pp. 966.  
Meeting Info.: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA October 23-28, 1999 Society for Neuroscience  
. ISSN: 0190-5295.

L34 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI The G protein-coupling profile of metabotropic **glutamate**

receptors, as determined with exogenous G proteins, is independent of their ligand recognition domain.

AB Metabotropic **glutamate** (mGlu), Ca<sup>2+</sup>-sensing, gamma-aminobutyric acid,, and a large number of pheromone receptors constitute a peculiar family of G protein-coupled receptors. They possess a large extracellular domain that has been proposed to constitute their ligand binding domain. The aim of the current study was to examine whether this large ligand binding domain had any influence on the G protein-coupling selectivity of the receptor, and vice versa. We chose mGlu receptors, which are classified into three groups according to their sequence homology and pharmacology, as representatives of this receptor family. To define a G protein-coupling profile for these receptors, we used a set of exogenous phospholipase C-activating G proteins in the same way that synthetic ligands are used to define agonist and antagonist pharmacological profiles. This set includes Galpha15, Galpha16, Galphaq, and chimeric Galphaq proteins with the last few amino acids of either Galpha12 (Galphaqi), Galphao (Galphaqo), or Galphaz (Galphaqz). Cotransfection of mGlu receptors with the G proteins and examination of their coupling to phospholipase C revealed that group I, II, and III receptors have distinct

G protein-coupling profiles. By swapping the extracellular domains of the most distantly related mGlu receptors (the rat group I mGlu1a and the Drosophila melanogaster group II DmGluA receptors), we show that the extracellular domain determines the agonist pharmacological profile and that this domain does not modify the G protein-coupling profile

determined

by the seven-transmembrane-domain region of mGlu receptors.

SO Molecular Pharmacology, (April, 1998) Vol. 53, No. 4, pp. 778-786.

ISSN: 0026-895X.

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L27	428 L22 AND L26
L28	311 L27 AND 1970-2000/PY
L29	33 G PROTEIN FUSION PROTEIN
L30	30 G PROTEIN CHIMER?
L31	61 L29 OR L30
L32	9 L22 AND L31
L33	4 L32 AND 1970-2000/PY
L34	4 DUP REM L33 (0 DUPLICATES REMOVED)

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